Position Paper

Epidemiology of Periodontal Diseases*

THIS PAPER WAS PREPARED BY THE RESEARCH, Science, and Therapy Committee of The American Academy of Periodontology and is intended for the information of the dental profession. It represents the position of the Academy in regard to the current state of knowledge about the epidemiology of periodontal diseases. Some reference is made to topics that are presented in *Tobacco Use and the Periodontal Patient and Periodontal Considerations in the HlV-Positive Patient,* though this paper takes an epidemiological perspective rather than one emphasizing patientcare. *J Periodontol 1996;67:935-945.*

INTRODUCTION

Epidemiology is the study of health and disease in populations, and of how these states are influenced by heredity, biology, physical environment, social environment, and personal behavior.Analytical epidemiology seeks to quantify the risk factors associated with a disease, which in turn can lead to theories of causation and produce hypotheses for its prevention and control. The essential features of epidemiology as a method of research, when compared to clinical research and case studies, are that 1) groups rather than individuals are the focus of study; and 2) persons with and without the condition of interest are included in studies which aim to quantify risk. On the other hand, clinical research, which can also study large groups of people, is confined to patients (i.e., those with the disease of interest). Epidemiological principles are also used in population surveys to determine prevalence and in clinical trials. More detaileddiscussions are available on the role that epidemiology has played in furthering our understanding of oral conditions,[1] and specifically of periodontal diseases. [2,3]

Advances in research over recent years have led to a fundamental change in the periodontal diseases model. "Model" in this context means a collection of theory on etiology and progression, based on research as far as possible. As recently as the mid-1960s, the prevailing model of periodontal diseases included these precepts: 1) all individuals were considered susceptible to severe periodontitis; 2) gingivitis progressed to periodontitis with consequent bone loss and tooth loss; and 3) susceptibility to periodontitis increased with age.[4-6] All of these aspects of the disease model from that time are now, at the very least, under strong challenge from the results of more recent research.

This review examines the epidemiology of periodontal diseases, concentrating on recent research in adult periodontitis. It will review current knowledge on prevalence, incidence, severity, and risk factors. Issues in the measurement of periodontitis are not specifically reviewed, though there is necessarily some consideration of how data are collected from population groups. The review does not directly address microbial infection and host response mechanisms, modes of disease progression (i.e., bursts or linear), nor the less common clinically recognized conditions such as rapidly progressive, prepubertal, refractory, and localized and generalized juvenile periodontitis.

The Prevalence of Periodontal Diseases

Prevalence is the number of cases of a disease in a designated population at a given point in time.[7] For reasonably common conditions like periodontal diseases, prevalence can be estimated from the results of national surveys conducted by the National Center for Health Statistics and the National Institute of Dental Research, with additional data from smaller-scale surveys of population samples or specific, non-representative groups. Any prevalence information must be interpreted in light of the population studied, the clinical examination conditions, and the training and experience of the examiners.

Perhaps the most fundamental change from the old model of periodontal diseases described earlier is that the belief in universal susceptibility has given way to the current view that only some 5% to 20% of any population suffers from severe generalized periodontitis, even though moderate disease affects a majority of adults.[8] This clustering of serious disease has been observed among well-treated patients [9] as well as in epidemiologic studies

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among a variety of populations.[10-14] Periodontitis is now seen as resulting from a complex interplay of bacterial infection and host response, often modified by behavorial factors.[15]

Any measure of prevalence of periodontitis is dependent on how the disease is defined, i.e., the case-definition of periodontitis. If the disease is defined as the identification of at least one site with clinical attachment loss (CAL) of 2 mm or more, around 80% of all adults are affected, and over 90% of those aged 55 to 64.[8] When the case-definition is at least one site with CAL of 4 mm or more, the prevalence in those aged 55 to 64 drops to 64%. When it is CAL of 7 mm or more, prevalence drops to below 10%. Making due allowance for the uncertainty with case-definitions, it is clear that mild to moderate periodontitis is so prevalent the milder forms are close to universal. The severe manifestations of the disease, meaning those that lead to tooth loss, or at least threaten it, are less prevalent. Even just these few data demonstrate that any prevalence data are of little value without the relevant case-definition and the age-group to which they apply.

Periodontal diseases have been part of the human condition for much of recorded history.[16] Trends in prevalence and severity of periodontal diseases, however, are not easy to ascertain because the philosophical basis for their measurement has changed with the disease model. Even so, national survey data for the United States suggest that gingivitis prevalence has declined over the last 30 years or so, and that while the prevalence of periodontitis has changed little, its severity has diminished.[17,18] Parallel improvements in periodontal health have been reported in Scandinavia.[19,20]

Gingivitis: Prevalence and Distribution

When assessed in populations, gingivitis is found in early childhood, and is more prevalent and severe in adolescence, after which prevalence tends to level off.[21] The prevalence of gingivitis among school children in the United States has ranged from 40% to 60% in national surveys. [22,23] In the national survey of employed adults in 1985-86, 47% of males aged 18 to 64 exhibited at least one site which bled on probing and females 39%.[8] The mean number of bleeding sites per person increased with age in males, but not in females. In the first national survey of adults in 1960-62, which judged gingivitis visually, some 85% of men and 79% of women were found to have some degree of gingivitis.[24] Even allowing for the differences in measurement techniques between the two surveys, there appears to have been an improvement in gingival health over that period.

Gingivitis is closely correlated with plaque deposits, a relationship long considered one of cause-and-effect. Studies on the natural history of periodontal diseases in Norway and Sri Lanka found no increase in prevalence and severity of gingivitis between the late teen years and age 40 in Norwegian professionals and students, among whom oral hygiene was excellent.[25] Among Sri Lankan tea workers, both the gingival condition and oral hygiene were poorer at all ages. Surveys among other third-world populations show that gingivitis, associated with extensive plaque and calculus deposits, is the norm among adults.[26 28]

It is generally believed that gingivitis has declined over recent years in the United States because of greater attention to oral hygiene as a part of personal grooming. The main interest in gingivitis today is whether it is a precursor to periodontitis, since research suggests that only some sites or patients with gingivitis go on to develop periodontitis.[29-31]

Measurement of Periodontitis

Periodontitis is defined as inflammation of the gingival tissues together with some loss of both the attachment of the periodontal ligament and bony support.[32] The standard clinical measures for periodontitis, apart from gingival bleeding and radiographic assessment of bone loss, are clinical attachment loss (CAL) and probing depth (PD). The standard protocol used today for measuring CAL and PD was first described over 35 years ago.[33] Various scaled indexes have been used in the past, but most are now considered of dubious validity and have thus been discarded. Although CAL, a measure of accumulated disease at a site rather than current activity, remains a diagnostic "gold standard" for periodontitis,[34] the absence of consensus on how best to use CAL and PD in a case-definition of periodontitis continues to hamper clinical and epidemiological research. Studies have measured CAL and PD on all teeth, all teeth in two quadrants, the worst teeth in each sextant, and selected index teeth. Measurements have been made on six, four, two, and one sites per tooth, and the most appropriate use of high-technology diagnostic equipment (e.g., computerized probes and DNA probes) continues to be debated. As one illustration of this problem, it has been suggested[35] that the 1985-86 National Survey of Oral Health in U.S. Employed Adults and Seniors[8] may have underestimated the national prevalence of periodontitis because it measured only two sites per tooth (mesiobuccal and midbuccal) in one maxillary and one mandibular quadrant. Furcation and lingual areas, the places where disease is considered most likely to develop, were not included in the survey protocol. This probably biased the disease estimates downward. (From the epidemiological perspective, another source of underestimation in the national surveys is likely to be the necessary sampling restrictions: the 18- to 64-year-olds were employees only, mostly from small-sized businesses, and the seniors were those who attended senior centers. This sample was quite probably biased toward individuals with better periodontal health.)

A case-definition for periodontitis needs to establish 1)

what depth of CAL at any one site constitutes evidence of disease processes; and 2) how many such sites need to be present to establish disease presence. The first issue also has to make allowance for examiner variation, which can confuse efforts to measure CAL progression. The standard deviation of repeated CAL measurements of the same site by an experienced examiner with a manual probe is around 0.8 mm,[36] so change in attachment level in a clinical study needs to be at least 2 mm (i.e., 2 to 3 times the standard deviation) before the investigators can be confident that they are seeing real change rather than measurement error.[37,38] CAL progression of at least 3 mm over a given time period has been the criterion for change in other studies.[39,40] In epidemiology, use of the 2 mm level to denote CAL in a cross-sectional study can be questioned because it is too common[8] to serve as a dis–ease threshold. When the CAL cutoff limit is raised to 4 mm, the distribution of affected individuals becomes sufficiently skewed to discriminate adequately between those with and without CAL of this extent,[10] but this level can be criticized as being too conservative. Regarding the extent of CAL in a mouth, the issue is how many sites need to be affected in order to give an accurate profile. An approach like the Extent and Severity Index,[41] in which "extent" refers to the number of teeth in the mouth with CAL of I mm or more, and "severity" is the mean CAL for those teeth, might be appropriate in some circumstances, although the CAL cutoff limit of 1 mm needs to be increased for the reasons discussed previously. Some consensus on age-related case-definitions for "serious" and "moderate" disease would also assist research. A number of recent studies have used their own case-definitions for "serious" disease, mostly based on combinations of CAL and PD or extent of bone loss,[12 14,20,42-45] but no uniformity has yet developed.

In addition to the problems that stem from uncertainties with case-definitions, the very meticulousness of the necessary clinical measurements required can inhibit epidemiological and clinical research in periodontitis. It would be a considerable advance if the clinical measures could be supported by a reasonably simple medical test which would permit categorization of subjects into those with and without active disease processes. No such test with the required attributes of sensitivity, specificity, and predictive values has yet emerged, though some, such as measures of the inflammatory mediator prostaglandin E_2 in gingival crevicular fluid,[46] hold promise and merit further development.

Incidence of Periodontitis

Only a few longitudinal studies on periodontitis have been conducted because of their inherent difficulties and expense. Epidemiological studies in the community are especially demanding, though clinical studies with pa–tients seeking treatment are difficult enough. Several longitudinal studies have confirmed the results of cross-sectionalstudies in identifying age as a risk factor for progression ofCAL,[47,48] though others[44,49,50] concluded that progression of CAL is more closely related to the extent of baseline CAL. In the Piedmont study in North Carolina, which for three years followed a population sample aged 65 to over 80, baseline findings were that CAL was much more extensive in study participants than it was in younger groups.[42]

Subsequent reports from the Piedmont study have contributed to our understanding of the natural history of periodontitis. Using 3 mm as a conservative estimate of CAL, it was found that African-Americans were more likely to experience CAL than whites, and about half of the whole group experienced at least onesite with 3 mm CAL over the first 18 months.[40] After three years of observations, it was found that about half of the group did not experience additional CAL at all, and only some 12% experienced CAL in each of the two 18-month periods.[51] It was also found that CAL during the first 18 months was related to subsequent CAL at the subject level though not at the individual site level, a finding which supports the episodic, randomized model of periodontitis. At the site level, past disease predicted subsequent CAL. An intriguing finding was that 58% of those with incident CAL developed this condition mostly from increasing probing depth, the remaining 42% mostly from recession.[52] The authors suggest that different disease processes may be at work here.

A 15-year longitudinal study of 480 tea workers in Sri Lanka demonstrated a wide range of susceptibility to periodontitis.[12] Thegroup studied had virtually no dental treatment, so the data reflected the natural history of periodontitis. Based on tooth loss and interproximal CAL, it was concluded that about 8% demonstrated rapid progression of periodontitis, some 81% showed moderate progression, and the remaining 11% showed no progression beyond gingivitis. In the rapid- and moderate-progression groups, periodontitis progressed with age (much more rapidly in the first group), whereas in the non-progressing group age was not a factor. This study demonstrated that CAL became severe over time only for a small group of susceptible individuals.

DETERMINANTS AND RISK FACTORS FOR PERIODONTITIS

A *risk factor* is an environmental exposure, aspect of be–havior, or an inherent characteristic which is associated with a disease. The association may or may not be causal,[7] though the use of the term often implies causality. The term *determinant* is often used synonymously with risk factor in the literature, but for clarity is best reserved for risk factors that cannot be modified. In the same vein, determinants are sometimes referred to as background factors in risk assessment models. The term *risk indicator*

describes plausible correlates of disease identified in cross-sectional studies, while risk factor is best applied to those correlates confirmed in longitudinal studies. Risk factor implies a modifiable condition. Risk indicators identified in cross-sectional studies are not always confirmed as risk factors in longitudinal studies.[3] *Risk marker* is used more in the predictive sense, a factor associated with increased probability of future disease but where causality is usually not implied.

Determinants

Age. The prevalence and severity of CAL is directly related to older age in cross-sectional surveys. In the 1985-86 national survey, the proportion of adults with at least one CAL site of 2 mm or more exceeded 70% even in adults aged 35 to 44 years, and was more than 90% in those aged 55 to 64.[8] The presence of at least one CAL site of 4 mm or more increased steadily from 13.8% of 25- to 34-year-olds to 53.6% of 55- to 64-year-olds. When the minimum CAL was set at 7 mm, there was again a steady increase with age from 3.8% of 35- to 44-year olds to 9.3% of 55- to 64-year-olds affected. PD is also related to age, though less directly than is CAL. The 1985-86 national survey found that pockets of 4 to 6 mm were present in 13.4% of all adults, and were more frequent in older age groups.[53] But pockets of 7 mm or more were found in only 0.6% of those examined, and were not related to age.

The long-term assumption that periodontitis is a disease of aging has been challenged in recent years[11,54-57] The current view sees the greater periodontal destruction in the elderly as reflecting lifetime disease accumulation rather than an age-specific condition. A relatively low prevalence of severe (as opposed to moderate) CAL among the elderly was first shown in Sweden,[58] and has since been demonstrated elsewhere. Surveys of older people in the United States, Canada, and Australia have found that CAL or PD of 6 mm or more was prevalent in some 15% to 30% of persons examined.[59-62] In all of these studies, CAL of 4 to 6 mm was common. Higher estimates of periodontal destruction came from a cross-sectional New England study of community-living elderly people.[63,64] The New England study was of persons aged 70 to 96, older than those seen in the 1985-86 national survey, and the results could reflect cohort effects (i.e., results specific to the generation studied and which may not be seen in subsequent generations). All of these reports agree that CAL increases with age, but most did not find extensive loss of function in the affected teeth.

Periodontitis frequently begins in youth and early adulthood, rather than in the older years, for some degree of CAL in youth is well documented in population studies.[65-72] It can be hypothesized that the more susceptible members of the population are those in whom periodontitis begins in youth. If that is so, then the relatively low prevalence of severe CAL. among many dentate elderly could be partly a survival phenomenon, meaning that those most susceptible to severe periodontitis have already lost teeth. The most rapid disease progression is seen in that relatively small number of persons in whom the disease starts young. Despite widespread assumptions, it is not yet clear whether this type of disease is associated with leukocyte defects; this issue has received virtually no epidemiological study.

It is uncommon for elderly people with reasonably intact dentitions to exhibit sudden bursts of periodontitis.[54] Tooth retention, good oral hygiene, and periodontal health (exhibited by little gingival inflammation and few deep pockets) are closely associated, regardless of age.[73,74] The New England studies have raised the issue of whether periodontal destruction becomes more severe among the oldest of the elderly, while the Piedmont studies have shown that levels of periodontal destruction among older persons are highly variable. A question that will have to be addressed, especially as the population ages and tooth retention increases, is what level of periodontal destruction among the elderly can be tolerated as compatible with an acceptable quality of life.

Gender. CAL of all levels of severity is generally more prevalent in males than in females. This has been a consistent finding in all national surveys in the United States since the first in 1960-62.[8,24,75] Males usually exhibit poorer oral hygiene than females, both calculus and soft plaque deposits.[8,73,76] In an Iowa study of dentate persons aged 65 or older, age was negatively related to the proportion of teeth classed as periodontally healthy in males, but positively in females.[77]

The reasons for these gender differences have not been explored in detail, but are thought to be related to poorer oral hygiene and dental-visit behavior among males than to any genetic factor. There are of course certain genderrelated temporary syndromes related to hormonal conditions, such as “pregnancy gingivitis,” as well as pubertal gingivitis which can affect children of both sexes. Women aged 50 to 64 receiving estrogen replacement therapy were found to have less gingival bleeding than controls, even after controlling for higher educational levels and lower plaque accumulations.[78]

Socioeconomic Status (SES). Levels of periodontal disease have historically been related to lower SES.2475 Gingivitis and poor oral hygiene are clearly related to lower SES, but the relationship between periodontitis and SES is less direct. The 1985-86 national survey[8] found that the prevalence of CAL at all levels of severity was not closely related to household income. CAL of 4 mm or more and 7 mm or more in at least one site were both closely correlated with educational levels, though the relation was much weaker with CAL of 2 mm or more because this measure was so common.

SES levels and gingival health is a function of better oral hygiene among the more educated, and a greater frequency of dental visits among the more dentally-aware and those with dental insurance (who are more likely to be white-collar employees; i.e., those with more education). While racial/ethnic differences in periodontal status have been demonstrated many times, it is still not clear whether these are true genetic differences or whether SES, a complex and multifaceted variable that can include a variety of cultural factors, is confounding these relationships.

Risk Factors

Plaque, microbiota, and oral hygiene. The relationship of oral hygiene to periodontitis is not as straightforward as that seen with gingivitis. The conclusion from most cross-sectional studies in populations with poor oral hygiene is that plaque and supragingival calculus accumulations correlate poorly with severe periodontitis.[12,13,26-28,79-83] On the other hand, a correlation between oral hygiene levels and periodontal diseases was found in a study of Norwegian 35-year-olds, but this study measured periodontal conditions by the Periodontal Treatment Needs System (PTNS), a forerunner of the Community Periodontal Index of Treatment Needs (CPITN), which weights gingival inflammation and calculus rather than CAL.[19] Another retrospective study found an association between plaque deposits and CAL over a 10-year period, though these results could have been biased by subject selection.[49] Subgingival calculus, as a separate entity from the supragingival variety, has not been studied epidemiologically.

Results from well-controlled studies have also found that quantity of plaque accumulation was, at best, only weakly correlated with periodontitis,[31,84-89] and predictive models have also been unable to identify plaque deposits as predictors of future CAL.[37,90] Clinical findings from the Karlstad studies in Sweden, however, concluded that CAL in susceptible adults can be halted almost completely when meticulous self-performed plaque control is combined with professional prophylaxis 3 to 6 times per year. The prophylaxis in these studies included sub- and supragingival scaling, and root planing.[91]

Studies using qualitative measures of plaque (i.e., microbiota), rather than just plaque quantity, have produced mixed results. Cross-sectional associations between putative periodontopathogens and clinical periodontitis have been reported,[92-93] and their presence in subgingival plaque samples from susceptible patients has predicted CAL over the short term.[44] The combination of calculus, gingival bleeding, and the presence of *Prevotella intermedia* provided the best explanation for CAL in a group of Navajo adolescents aged 14 to 19.[94] On the other hand, microbiota could not predict the development or progression of periodontitis in clinical longitudinal studies for up to three years.[95-97]

Epidemiological data show that while there is generally more CAL in third-world populations than in the western world, the deficient oral hygiene and consequent gingivitis in such populations does not always progress to periodontitis.[12,13,26-28] These findings emphasize the role of the host response in the development of clinical periodontitis, so it is not surprising that the identification of an infection by itself does not predict periodontitis very well. The best predictor of future CAL still appears to be past disease added to age,[44,98] and the very presence of past disease indicates a susceptible host. Prediction of future disease, either in the person or at a site, is still an inexact procedure.

Tobacco. An analysis of data from the 1971-75 National Health and Nutrition Examination Survey in the United States (NHANES I) showed a clear association between smoking and periodontal diseases, independent of oral hygiene, age, or any other factor.[99] The evidence to identify smoking as a risk factor for peridontitis has continued to mount since then[47,85,,86 100,101] and assessments of randomly-chosen patient groupings invariably show a higher prevalence of periodontitis among smokers.[102-104]

Experimental studies have shown that there is no difference between smokers and non-smokers in amounts of plaque accumulation,[102,105] nor in the prevalence of the principal bacteria which are considered pathogenic for periodontitis.[106,107] What smoking appears to do is suppress the vascular reaction which follows gingivitis. In experimental plaque-induced gingivitis, despite the rate of plaque accumulation being equal in smokers and non-smokers, the increase in gingival vascularity in smokers was only half of that seen in the non-smokers.[108] In effect, this is a masking effect on the signs of inflammation.[109] This finding might explain the results of the one epide–miological study in which smoking was not found to be associated with periodontal diseases.[110] In this study of a representative sample of the Finnish population, the periodontal assessment was by means of the previously mentioned PTNS, which weights gingival inflammation and plaque deposits. The effect of smoking in suppressing the signs of gingival inflammation could have led to the negative findings.

There is also some evidence that smoking is associated with osteoporosis, the role of which in alveolar bone loss is currently under study.[111] A study of denture require–ments among postmenopausal women found that twice as many smokers as non-smokers required dentures after age 50, and that the lowest need was among non-osteoporotic non-smokers.[112] Smoking may also be a factor in the as–sociation between refractory periodontitis and a polymorphonuclear leukocyte defect in the peripheral blood.[113] The risk of periodontitis attributable to tobacco, compared to its non-use, is in the order of 2.5 to 6.0 or even higher.[109] The evidence is clear that smoking is a major risk factor for periodontitis.

Systemic Conditions

Reviews of the role of systemic predisposition to periodontitis have concluded that there is little firm evidence for a relationship, apart from syndromes associated with PMN defects (e.g., Chediak-Higashi syndrome), Down's syndrome, Papillon-Lefévre syndrome, and the rare condition of Ehlers-Danlos syndrome.[114,115] These reviews also concluded, however, that insulin-dependent diabetes and acquired immunodeficiency syndrome (AIDS) may exacerbate the effects of existing disease.

Diabetes. Both type I (insulin-dependent diabetes mellitus, or IDDM, formerly referred to as early-onset diabetes) and type II diabetes (non-insulin-dependent diabetes mellitus, NIDDM, formerly referred to as adult-onset diabetes) are risk factors for periodontitis. Younger adult patients with IDDM, especially those in whom the disease is of long duration, have been found to have more gingivitis and more deep pockets than non-diabetics.[116-119] Among patients of similar age with long-duration IDDM and similar plaque levels, those with poorer metabolic control had greater CAL and alveolar bone loss than did those with better control.[120-122] Periodontitis also progresses more rapidly in poorly-controlled diabetics,[121] and early age of onset of diabetes also is seen as a risk factor for more severe disease.[123] Poorly-controlled diabetics have also been found to exhibit higher levels of the enzyme beta-glucuronidase in their gingival crevicular fluid than do well-controlled patients.[124]

The most extensive studies among NIDDM patients have been among the Gila River community in Arizona, where NIDDM prevalence is high. NIDDM patients had substantially greater CAL, loss of alveolar bone, and tooth loss.[48,125] With age, sex, and oral hygiene all controlled, the increased risk of developing destructive periodontitis as a result of NIDDM was 2.81 for CAL and 3.43 for bone loss.[126] These degrees of risk are similar to the odds ratio of 2.32 for severe CAL in diabetics (variety not specified) compared to non-diabetics in an adult population in upstate New York.[86] Metabolic control is a prime factor in maintaining periodontal health among NIDDM patients,[127-129] and there is some evidence for the converse, namely that periodontal health may affect diabetic control.[130]

A study of quantitative and qualitative aspects of microflora in both type I and type II diabetics revealed no notable differences between diabetics and non-diabetics.[131] Tests of subgingival plaque samples from participants in the Gila River studies suggest that diabetics react differently to the putative pathogens than do non-diabetics, though the significance of these findings awaits further study.[132] Other suggested mechanisms by which diabetes may contribute to periodontitis include vascular changes, PMN dysfunction, abnormal collagen synthesis, and genetic predisposition.[133-135] While the mechanism by which diabetes exacerbates periodontal destruction is still not fully understood, periodontitis can be considered a complication of both types of diabetes. Poor diabetic control exacerbates the risk even further.

HIV Infection. Although many studies on HIV infection and periodontitis were not rigorously designed,[136] HIV infection is considered to be a risk factor for periodontitis. Issues of caring for the HIV-infected patient have been considered in the Academy paper *Periodontal Considerations in the HIV-Positive Patient.*[137]

Initial research was limited to cross-sectional studies of convenience samples of homosexual men.[138,139] The microbiology of periodontitis in HIV-infected persons, relative to those not infected, is not clear, for both little difference[140-142] and significant differences[143] have been reported. None of these studies reported on the immunosuppression status of the subjects, though a later assessment of HIV-infected patients found the occurrence of necrotizing ulcerative periodontitis to be related to decreasing CD4+ counts.[144] Reports on the occurrence of yeasts in subgingival flora of HIV-positive patients do not give a clear picture.[143,145]

Clinical studies of persons with HIV infection are limited. Some are limited to descriptive studies of groups of patients without controls.[146,147] One comparison between men at different stages of HIV disease found little difference in terms of periodontal health,[148] a finding which seems at odds with earlier reports of greater severity of periodontitis in advanced HIV disease.

Several studies have examined the periodontal condition of patients taking part in clinical trials for the drug zidovudine (AZT). One reported the periodontal health of patients in the early stages of HIV disease to be generally good,[149] and a longitudinal study of 30 HIV-infected patients found a greater progression of periodontitis over 18 months in this group in comparison to 10 HIV-negative controls.[150] In neither study, however, was there an analysis of periodontal disease in the zidovudine patients compared to the others.

In a follow-up of 114 homosexual or bisexual men conducted over a period of 20 months, periodontal changes were found to be related to HIV-I serostatus, immune status, age, and plaque deposits. The risk of CAL of 3 mm or more over the 20 months was 6.16 times higher in the more immunosuppressed patients (CD4+ counts less than 200) than in the less immunosuppressed patients, and this finding was more pronounced in older subjects.[151] Seropositive patients showed a more sensitive reaction to plaque than did the seronegative patients. This well-conducted study concluded that immunosuppression, especially in combination with older age, was a risk factor for progression of CAL, and that seropositivity, independent of immune status, was a risk factor for gingivitis.

In a cross-sectional study of 230 HIV-infected military personnel, however, the relation between periodontal health and immune status was less clear.[152] A detailed

follow-up on 474 patients from the same population, aged 18 to 49, found CAL of 5 mm or more in 20% of the patients. In this group, which was nearly 85% male, neither the clinical stage of the disease nor the CD4+ counts were good independent predictors of severe CAL when other significantly associated variables (e.g., tobacco use) were accounted for.[153] Perhaps the most intriguing finding from this study is that severity of periodontitis may be related to use of the drug zidovudine, possibly through interference with neutrophil function.

Other Systemic Conditions. There is evidence that periodontal infections are associated with some serious systemic illnesses. While a case of septic pulmonary embolism involving *Streptococcus intermedius* from periodontal lesions has been reported,[154] the role of periodon–titis in cardiovascular conditions has attracted more attention. One study analyzed clinical periodontal data from the NHANES I survey of 1971-75, and matched it with cardiovascular data obtained from death certificates, personal interviews, and records from hospitals and nursing homes. The report concluded that persons with periodon–titis at baseline had a 25% greater risk of subsequent cor–onary heart disease than did those without periodontitis.[155] The risk was especially strong for men under age 50 at baseline, for whom the relative risk was 1.72. This evidence is provocative, though by itself not conclusive because periodontal data was obtained by the now-defunct Periodontal Index, and because periodontal conditions were measured only at baseline. This analysis also found that periodontitis and poor oral hygiene were more strongly associated with total mortality than with coro–nary heart disease, which could indicate that neglect of oral health is more an indicator of poor health practices in general than a causal factor. Associations of varying strength between cardiovascular disease and periodontitis have been reported from different populations, [156-159] evidence which is consistent though not sufficient by itself for causality. Evidence for a causal link, however, can be found in an animal study in which rats with both aortic valve vegetations (induced by catheter) and with perio–dontitis were subjected to tooth extractions. A subsequent bsvyrtr,os was observed, and three days later 90% of the rats had endocarditis.[160]

The infections which characterize periodontitis have been associated with a variety of systemic conditions;[161] *Actinobacillus actinomycetemcomitans* in particular has been involved with endocarditis and brain absesses.[162] It has been suggested that specific properties of bacteria, such as ability to adhere to damaged heart valves, might be more important than simply the numbers which enter the bloodstream in a bacterial shower.[163]

It is interesting to reflect that what is being discussed here is essentially the "focal infection" issue, first raised in 1911,[164] and which subsequently became the underlying rationale for mass extractions over subsequent decades. The focal infection theory faded in the 1950s, and now may be re-emerging. The link between periodontitis and cardiovascular disease, and perhaps other conditions as well, requires further research in well-controlled epidemiological studies in order to determine the extent of any causal link.

SUMMARY

1. The prevalence of periodontitis has declined little over a generation or so but its severity probably has. Data on the prevalence of periodontitis are dependent on how the disease is defined and the age group from which they were taken.

2. Some 5% to 20% of the population suffer from severe, generalized periodontitis, though mild-to-moderate periodontitis affects a majority of adults. The severest forms of periodontitis appear to be evident in teenage and early adult years rather than the elderly.

3. There is no consensus on case-definitions for mild, moderate, and severe periodontitis. Research would benefit from uniform definitions that could be used in human studies to express prevalence, incidence, and risk assessment.

4. No clear picture of disease incidence has yet emerged, though the most consistent predictor of future disease in a particular site is previous disease.

5. Risk factors for periodontitis include smoking and several systemic diseases. The role of plaque and supragingival calculus deposits as risk factors is still not well demonstrated.

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